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Contagious Bovine Pleuropneumonia in Ethiopia (Review Article)

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Abstract: Contagious bovine pleuropneumonia is a disease of cattle caused by *Mycoplasma mycoidessubsp. mycoidessmall* colonies. The disease is characterized by a relatively long incubation period and a highly variable clinical course. Recovered animals may harbour the infection in lung sequestra: necrotic areas of lung tissue separated from the surrounding normal tissue by a fibrous capsule. Contagious bovine Pleuropneumonia is current disease of major concern throughout sub-Saharan Africa. The principal route of infection is by the inhalation of infective droplets from animals active or carrier cases of the disease. An essential part of the pathogenesis of the disease is thrombosis in the pulmonary vessels, probably prior to the development of pneumonic lesions. It is manifested by anorexia, fever and respiratory signs such as dyspnoea, polypnoea, cough and nasal discharges. Diagnosis depends on the isolation of an etiological agent. The common methods used for the diagnosis of the disease are complement fixation test and enzyme linked immune sorbent assays. It is considered to be a disease of economic importance. The disease is endemic in Ethiopia. The major control method practiced in Ethiopia is vaccination. The main problems for control or eradication are the uncontrolled movements of animals and the frequent occurrence of sub-acute or subclinical infections and the persistence of chronic carriers after the clinical phase. Therefore, adequate control strategic measures should be implemented for eradication of the disease such as test and slaughter, stamping out, quarantine and vaccination.

Key words: CBPP • Control Strategies • Diagnosis • Epidemiology • Ethiopia *Mycoplasma Mycoides*

INTRODUCTION

Contagious bovine pleuropneumonia (CBPP) is a contagious disease of cattle caused by *Mycoplasma mycoidessubspecies mycoides* Small colonies (*MmmSC*) [1]. It has been known to occur in Europe since the 16th century but it gained a world-wide distribution only during the second half of the 19th century because of increased international trade in live cattle. It was eradicated from many countries by the beginning of the 20th century through stamping-out policies. However, the disease persists in many parts of Africa. The situation in Asia is unclear, but historically it was thought the disease was introduced into Europe from Asia in 19th century and that wars of the 18 and 19th century resulted in its spread throughout the continent. From Europe it was taken to the rest of the world; South America is the only continent that

has never experienced the disease [2]. There has been no reported outbreak in Europe since 1999. In natural conditions, *MmmSC* affects only the ruminants of the Bosgenus, i.e. mainly bovine and zebu cattle [1].

The disease is an OIE-notifiable disease and was included among the former list “A” diseases [3, 4]. It is in a prominent cattle disease in Africa, where outbreaks of the disease reported from 20 countries 2006, with the highest number of cases in Ethiopia, Angola and Cameroon [5]. CBPP has been eradicated in Australia, Europe, Asian and America through the application of restrictions to the movement of cattle, as well as test and slaughter policies combined with compensation for livestock keepers. Such policies are difficult to apply in most African countries because of pastoralism, lack of economic resources and fragmented veterinary services [6, 7]. As a result, the disease remains endemic in Africa

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particularly in tropical and subtropical regions (West, central, east and parts of southern Africa) of the continent [6, 8]. The disease has serious implications for food security and peoples' livelihoods in affected countries [8].

Contagious bovine pleuropneumonia is manifested by anorexia, fever and signs polypnoea, cough and nasal discharges. In the case of acute outbreaks under experimental conditions, the mortality rate may be as high as 50% in the absence of antibiotic treatment. When an outbreak first occurs in an area, the mortality will be high but is often lower in the field following the primary outbreak. Clinical signs are not always evident; sub-acute or asymptomatic forms occur frequently as the clinical signs in affected animals subside with partial recovery. In this case their lungs show typical encapsulated lesions called 'Sequestra'. These animals may be responsible for unnoticed persistence of the infection in a herd or a region and play an important role in the epidemiology of the disease [1, 9, 10].

Its transmission occurs from direct and repeated contacts between sick and healthy animals (Naïve one). There is no evidence of transmission through fomites as *MmmSC* does not persist in the environment. The principal route of infection is by the inhalation of infective droplets from animals active or carrier cases of the disease. Outbreaks tend to be more extensive in housed and in those in transit by train and on foot [9]. Factors such as extremes of age, stress and concurrent infections may predispose to tissue invasion [11]. In most continents, control strategies are based on the early detection of outbreaks, control of animal movements and a stamping-out policy. In Africa control of the disease is based on vaccination campaigns using attenuated *MmmSC* strains such as T1/44 or T1sr [1, 9]. It is considered to be a disease of economic importance because of its high mortality rate, production loss, increased production cost due to cost of, disease control, loss of weight and working ability, delayed marketing, reduced fertility, loss due to quarantine, loss of cattle trade and reduced investment in livestock production [3,9].

Although the use of antibiotics is theoretically prohibited, they are widely applied in the field. The consequences of these antibiotic treatments in terms of clinical efficacy, emergence of resistant strains and persistence of chronic carriers have not been evaluated yet. However, recent work has shown that antibiotic treatment of cattle may greatly reduce the transmission to healthy contacts but this requires treatment of all affected

cattle in a group [12]. An overview of the current state of techniques available for the diagnosis of CBPP clearly demonstrates that recent advances in the study of immunology and molecular biology have and will continue to open avenues for improved CBPP diagnosis. The tools currently available for CBPP diagnosis include clinical signs, pathologic lesions, (Pleurisy, lung hepatization), identification and isolation of the agent, immunoblotting, serology and PCR techniques [13].

Ethiopia is a tropical African country in which mobile pastoralism is dominant in the arid and semi-arid areas in the eastern, northeastern and southeastern parts of the country [14]. Currently, CBPP is one of the most important cattle diseases and impediments to livestock development in Ethiopia [8, 15]. Studies undertaken on CBPP so far revealed the existence of the disease in different parts of the country with prevalence that varies from 4.3 % in Jijiga [16] to 96 % in Western Gojjam [17]. The cattle population at risk of CBPP and livestock production systems in CBPP endemic and epidemic zones of Ethiopia is estimated to be a total of 13,325,700 heads of cattle [18]. Although the disease is endemic in the country and brings a high economic loss in the livestock industry, there is not enough information regarding its distribution and control in livestock industry as a priority disease in the country [19].

Therefore the objectives of this review paper are:

- To review the epidemiology of CBPP in Ethiopia
- To highlight diagnostic techniques of the disease
- To indicate control strategies of contagious bovine pleura pneumonia.

Contagious Bovine Pleuropneumonia: The disease: Contagious bovine pleuropneumonia (CBPP) is an acute, sub-acute or chronic respiratory disease of cattle caused by a *Mycoplasma* called *Mycoplasma mycoides* subspecies *mycoides* (Bovine biotype) SC (Small colony) [20]. It is a serious threat and obstacle to livestock production and development in Sub-Saharan Africa, some Asian countries and still occurring in some European countries. Once introduced to a new area, initial losses can be very high and its eradication is very difficult requiring major expenditure for control. CBPP is an economically important and highly infectious septicaemia characterized by localization in the lungs and pleura [10].

It causes a respiratory disease that ranges from a persistent, sub-clinical infection to an acute, sometimes fatal disease. Anorexia, fever and respiratory signs, such as dyspnoea, polypnoea, cough and nasal discharges, are the main manifestation of CBPP. The main problems for control or eradication are the frequent occurrence of sub-acute or asymptomatic infections and the persistence of chronic carriers after the clinical phase [20]. However, clinical signs are not always evident and could be confounded with other respiratory disease symptoms. Sub-acute or asymptomatic forms occur frequently and serve as a source for maintaining and spreading infection in the herd. Most infections are limited to the respiratory tract, although arthritis occurs in calves. Sequestra and chronic cases are possible but still remains debated the fact that these cases might be infectious in all cases and time (Lungersas a new source of infection) [21].

Etiology: The *Mycoplasmas* (*Mollicutes*), formerly called PPLO (Pleuropneumonia-like organisms), are non-sporulating, Gram-negative, non-motile bacteria, which do not possess a determined shape of the cell. The *Mollicutes* are members of the order *Mycoplasmatales* and class *Mollicutes* (*Softskin*) and they are the smallest of the free-living prokaryotes. *Mollicutes* is the correct term to use when collectively referring to members in this order; however, the trivial name *mycoplasma(s)* is also used for this purpose [2].

There are no internal membrane structures and no cell wall external to the plasma membrane; however, many strains possess surface structures equivalent to a capsule. With the exception of *Acholeplasmas*, *Mycoplasmas* depend on a supply of intact cholesterol, which they incorporate into the membrane, creating sufficient osmotic stability for survival under normal physiological conditions. The *Acholeplasmas* synthesize carotenol as a substitute for cholesterol, but will incorporate cholesterol if it is provided. Their polymorphism is the consequence of the missing cell wall. *Mycoplasmas* are devoid of not only cell walls but also lack the genetic capacity to produce one, which also renders the completely resistant to β -lactam and other cell-wall active drugs [22, 23]. Due to their small size (0.1-0.3 μ m) and their polymorphism, they are able to pass through the usual bacteriological filters (0.1-0.3 μ m). Cell shapes include spherical, pear shaped, spiral shaped and filamentous forms. Cell sometimes appear as chains and beads, the result of a synchronized genomic replication and cell division. *Mollicutes* stain poorly with

Gram stain method, although they are classified as gram negative. The preferred stains are; Giemsa, Castaneda, Dienes and methylene blue [21].

Members of the *Mollicutes* infect a wide range of animal species and human being. Infections range from sub-clinical to severely debilitating and sometimes fatal disease. Clinical manifestations include respiratory and uro-genital tract infections, arthritis, mastitis and septicaemia. Most pathogenic species exhibit a high degree of host specificity. *Mycoplasmas* are unique in microbiology because of their extremely small size and their growth on complex but cell-free media. Members of the *M. mycoides* group, *M. capricolum* group and Leach's group 7 form the so-called *M. mycoides* cluster, which consists of six *Mycoplasma* species, subspecies or groups of strains, originating from bovines and goats [24].

These six *Mycoplasmas* share serological and genetic characteristics and this causes taxonomic and diagnostic problems. In natural conditions, *Mycoplasma mycoides subspecies mycoides Small Colony type* (MmSC) affects only the ruminants of the *Bos* genus (Mainly bovine). Two types of *Mycoplasmas* are recognized: large colony (LC) and small colony (SC). They cannot be differentiated serologically but are different morphologically, culturally and in their pathogenicity and can be distinguished through mouse protection tests. Large colony types occur almost exclusively in goats, rarely in sheep while SC types cause CBPP in cattle. *Mycoplasma mycoides subspecies mycoides LC* also cause mastitis, arthritis and, occasionally, Contagious Caprine Pleuropneumonia and a fatal systemic disease in goats. They can be maintained readily in special culture media and in embryonated hens' eggs [24].

Epidemiology: Host range-Contagious bovine pleuropneumonia is predominantly the disease of the genus *Bos*; both bovine and zebu cattle are naturally infected. There are many reported breed differences with respect to susceptibility. In general, European breeds are tends to be more susceptible than indigenous African breeds [25]. Only cattle and water buffalo have been infected under experimental conditions [2]. There does seem to be some age resistance, animals less than three years of age are less resistant to experimental challenges [26].

Geographical distribution-Contagious bovine pleuropneumonia is endemic in parts of Africa, Middle East, Asia and sporadic outbreaks in some European countries [27]. It is a problem of in parts of Asia,

especially India, China. Periodically, CBPP occurs in Europe and out breaks within the last decades have occurred in Spain, Portugal and Italy. Contagious bovine pleuropneumonia was eradicated from the USA in the nineteenth century. It is of historical interest that the Bureau of Animal industries, which is the fore runner of the USDA'S Animals and Plants Health inspection service, was formed in 1884 specifically to eradicate CBPP. The USA was declared free of the disease only nine years later in 1993. Currently, CBPP is not present in the western hemisphere [9]. Methods of transmission-Normally transmissions are by droplet infection from actively infected animals to susceptible animals in close proximity [2]. Outbreaks usually occur as the result of movement of infected animals into a naïve herd. It is widely believed that the recovered animals harboring infectious organisms, within a pulmonary sequestrum, may become active shedders when stressed. Cattle may be exposed to infections for a period of up to 8 months before the disease become established and this necessitates a long period of quarantine before a herd can be declared to be free of the disease. Some inanimate objects such as placenta and urine can also remain infective for long periods; but this means of transmission is not general thought to be a problem [9, 28].

Incubation period-The time from natural exposure to overt signs of disease is variable but generally quite long. It has been shown that healthy animals placed in CBPP infected herd may begin showing signs of the disease 20 to 123 days. Experimentally subsequent to installation of a large quantity of infective materials at the trachea, bifurcation, the incubation period is 2 to 3 weeks [9].

Mortality and Morbidity-The attack rate with CBPP is variable. With increased confinement of animals, morbidity rises. The mortality with CBPP is quite varied and ranges from 10 to 70% in various outbreaks [2].

Source of infection-The primary source of most of the pathogenic mollicutes is the host that is infected with the agent [29]. The focus of infection is often provided by recovered carrier animals in which a pulmonary sequestrum preserves a potential source of organisms for periods as long as 3 years. For many, it was thought that condition of stress due to starvation, exhaustions or intercurrent can cause the sequestrum to break down and convert the animal in to an active case. Experimental evidence throws some doubt on this explanation, but droplet infection is usually associated with a donar lesion in the lung [9].

Risk Factors -Animal: Contagious Bovine Pleuropneumonia occurs only in cattle; rare natural cases have been observed in buffalo, yak, bison, reindeer and antelopes and the disease has been produced experimentally in captive Africa buffalo and white tailed deer [9]. A strong immunity develops after an attack of the natural disease in cattle and vaccination plays an important part in control. The lack of a cell wall and endotoxins may enable mycoplasmas to colonize the animal without inducing an immune response and the predilection for the mucosal membranes may also limit the humoral response [1, 9].

Management: The occurrence and incidence of CBPP is influenced by management system, disease control policies and regulation of the country, knowledge of the disease by farmers, veterinarians and livestock field officers. The diagnosis capabilities of veterinary laboratory, disease surveillance and monitoring system, adequacy vaccination programs, government budget allocated to control programs, desires of cattle owners and traders to control the disease are critically important manegment factors, which influence the effectiveness of controlling disease in a country [9].

Pathogen: *Mycoplasma mycoides* subspecies *mycoides* is sensitive to all environment influences, including disinfectants, heat and dry; do not ordinarily survive outside the animal body for more than a few hours. Restriction enzyme analysis of strains of the organism found that European strains have different patterns than African strains. The organism can be grouped into two major, epidemiologically distinct, clusters. One cluster contains strains isolated from different European countries since 1980 and second cluster contains African and Australian strains collected over the last 50 years. The current European strain lack a substantial segment of genetic information which may have occurred by deletion events. A variety of potential virulence factors have been identified, including genes of encoding putative variables, surface proteins, enzymes and transport proteins responsible for the production H_2O_2 and the capsule which is thought to have toxic effect on the animal. Molecular epidemiology of CBPP by multilocus sequence analysis of MmmSC strains found a clear distinction between European and African strains. This indicates that the CBPP outbreaks which occurred in European were not introduction from Africa and confirms true re-emergence.

The last strains isolated from an epidemic are usually of lower virulence than the first strains. Generally, strains are most virulent when first isolated and lose their virulence after subculture [1,9].

Pathogenesis: Contagious bovine pleuropneumonia is typical example of multi-factorial diseases, where factors such as intercurrent infections, crowding, inclement climatic conditions, age, genetic constitution and stress from transportation, handling and experimentation are important determinants of the final outcome of infection. An essential part of the pathogenesis of the disease is thrombosis in the pulmonary vessels, probably prior to the development of pneumonic lesions. The mechanism of development of the thrombosis is not well understood, but is considered, at least in part, mediated through induction of cytokines [30]. Contagious bovine pleuropneumonia is lobar variety of pneumonia in which the inter-lobular septa are dilated and prominent due to a great out pouring of plasma and fibrin in to them and this dilated septa that give the “Marbling” effect to the lung in these areas [10].

Bronchitis, bronchiolitis and alveolitis with predominantly neutrophils and mononuclear cellular response constitute the very early inflammation in *Mycoplasma pneumonia*. Contagious bovine pleuropneumonia is characterized by substantial unilateral pulmonary necrosis, sometimes sequestration and marked serosanguinous fluid accumulation in interstitial and pleura [31]. Vasculitis appears to be an important component of the pathological changes in this disease, explaining the marked exudation and pleurisy. Thrombosis can explain ischemic necrosis and infarcts of the lung. Death results from anoxia and presumably from toxemia [21]. There are various substances produced by the Mollicutes, which are potentially important in disease pathogenesis. Peroxide and super-oxide production may be important in disruption of host cell integrity [27].

Mycoplasma phospholipases are potentially important in pneumonia for they may reduce surface tension of the alveolar surfactants, thus resulting in atelectasis. A galactan polymer in *M. mycoides ssp. mycoides* has been shown to modulate the immune response and promote dissemination [31].

Clinical signs-There is considerable variation in the severity of clinical disease from hyper acute, acute, sub-acute to chronic form Radiostits *et al.* [9].

Hyper Acute Forms: The clinical signs observed in the hyper acute form are much accelerated. Affected animals may die within a week exhibiting classical respiratory signs. In fatal cases, death occurs after a variable course of from several days to 3 weeks [29].

Acute Forms: The early stages of CBPP are indistinguishable from any severe pneumonia with pleurisy. Animals show dullness, anorexia and irregular rumination with moderate fever and may show signs of respiratory disease. Coughing is usually persistent and is slight or dry. Sometimes fever goes up to 40 – 42°C and the animal prostrates with difficulty of movement. As the typical lung lesions develop, the signs become more pronounced with increased frequency of coughing and the animal becomes prostrate or stands with the back arched, head extended and elbows abducted. While classical respiratory signs may be evident in calves, articular localization of the causative agent with attendant arthritis usually predominates [2, 9].

Subacute Forms: Signs may be limited to a slight cough only noticeable when the animal is exercised. Cattle that recover naturally are extremely weak and emaciated. Many infected animals develop chronic or milder forms of the disease, which may be either symptomless or associated with only a slight temporary rise in body temperature and some loss of condition. Recovered animals may be clinically normal but in some, an inactive sequestrum forms in the lung, with a necrotic centre of sufficient size to produce a toxemia causing unthriftiness, a chronic cough and mild respiratory distress on exercise. The length of the incubation period depends upon the volume of the infective dose, the virulence of the strain and the immune state of the animal and it can last from a few days up to several months (In occasional instance up to 6 months) [31].

Depending on the resistance level of the animal and the intensity of exposure, the disease takes an hyper-acute, acute to chronic, or the acute course is sometimes followed by a chronic stage which may last for 2 to 3 years (Lunger) as a latent phase of the disease. The hyper acute form, involving up to 10 percent of infected animals, may be observed at the onset of an outbreak; death is sudden and is often not accompanied by any other signs. The acute form is observed in approximately 20 per cent of the diseased animals. The course is 5 to 7 days [21].

The earliest signs are a sudden onset of fever to 40°C or more and, in milking cows, a drop in milk yield, anorexia and cessation of rumination. There is severe depression and the animals stand apart or lag behind a traveling group and stop eating. The clinical symptoms start with the characteristics short, dry cough, which becomes more and more painful. Later, the cough usually becomes more severe; the animals shows signs of pain, standing with arched back and extension of the head and neck forwards and downwards, increased grunting respiration, salivation and nasal discharge. At this stage one could try to get sample of thoracic fluid from the chest by tapping before any fibrin is formed that would hamper the sampling (And auscultation of the lung is possible at this stage to identify formation of liquid [31].

Pathology-Gross Pathology: In acute CBPP, there is a severe fibrinous pneumonia with copious pleural exudates. The latter is a striking feature and there may be up to 30 liters' of yellow exudates, containing clots, in the chest cavity. One or both lungs may be partially or completely consolidated, giving a characteristic marbled appearance. Affected areas are swollen, vary from pink to dark red, have a moderately firm consistency and exude clear fluid and sometimes blood from cut surfaces. The interlobular septa are grossly thickened. Pleural surfaces over affected areas are thickened, grey. Nature of the disease is too red and is often covered by friable, yellow fibrin. Local lymph nodes are enlarged, edematous and may contain areas of necrosis [24, 32]. In chronic cases, necrotic lung tissue becomes encapsulated to form a sequestrum of 1 to 20 cm diameter. The tissue within the sequestrum [plural = sequestra] tends to retain much of the architecture of the acute lesion, but may eventually become calcified or liquefied. The lesion may either break open to release viable mycoplasmas or be resorbed. Pleural adhesions are commonly found in chronic cases [2,9].

Histopathology: Microscopically, the earliest pulmonary lesions consist of foci of catarrhal bronchiolitis, with distension of the lymphatic in the interlobular septa and thickened alveolar walls. At the same time, or soon after, blood vessels and lymphatic become thrombosis and alveoli are filled with fluid and cells (Alveolar macrophages and sometimes polymorphonuclear leucocytes). There is proliferation of the cells in lymphatic follicles and an increase in the population of mononuclear

cells around bronchioles. There is also lymphatic edema, with distension of sub pleural lymphatics. Necrosis can occur early and tends to have a lobular distribution. It is often demarcated from living tissue by a zone of leucocytes and nuclear debris [1, 24, 33].

A connective tissue capsule develops rapidly, but the necrotic material may persist for many months. Resolution of the pneumonia is by slow connective tissue replacement of damaged tissue. This starts around blood vessels. A layer of mononuclear cells borders the connective tissue on the necrotic side and connective tissue gradually moves in to replace the dead tissue [33].

Diagnosis and Diagnostic Techniques- The diagnosis of CBPP is based on a history of contact with infected animals, clinical findings, immuno-diagnosis tests, necropsy findings and cultural examination [9, 24].

Identification of the Agent: The causal organism can be isolated from samples taken either from live animals or at necropsy. Samples taken from live animals are nasal swabs or nasal discharges, broncho-alveolar lavage or transtracheal washing and pleural fluid collected aseptically by puncture made in the lower part of the thoracic cavity between the seventh and eighth ribs. Blood may also be cultured [1, 31].

Samples taken at necropsy are lungs with lesions, pleural fluid ('Lymph'), lymph nodes of the broncho-pulmonary tract and synovial fluid from those animals with arthritis. The samples should be collected from lesions at the interface between diseased and normal tissue. The agent can be detected by culture, nucleic acid methods and immunological tests described below. Bacteriological identification of the agent is more complex and can be done by biochemical tests, nucleic acid recognition methods and immunological methods. These methods are described here in general terms; however, it is recommended that the definitive identification be done by an OIE Reference Laboratory. The presence of pathogens varies greatly with the stage of development of the lesions and a negative result is not conclusive, particularly after treatment with an antibiotic. When dispatching samples to the laboratory, it is advisable to use a transport medium that will protect the mycoplasmas and prevent proliferation of other bacteria (heart-infusion broth without peptone and glucose, 10% yeast extract, 20% serum, 0.3% agar, 500 International Units [IU]/ml penicillin, thallium acetate 0.2 g/litre). The samples must be kept cool at 4°C if stored for a few days or frozen at or

below -20°C for a longer period. For laboratory-to-laboratory transfer, lung fragments or pleural fluid can also be freeze-dried [1].

Culture: *Mycoplasma mycoides* subspecies *mycoides* Small Colonies needs appropriate media to grow [1]. But it is not intrinsically difficult to grow, unlike other fastidious Mycoplasmas such as one causing CCPP, but requires a fully functioning bacteriological laboratory with access to special Mycoplasmas media [5]. In attempting isolation, 2-3 blind passages may be required. Many attempts to isolate fail because the organism is labile, is often present in small quantities and is demanding in its growth requirements. The media should contain a basic medium (Such as heart-infusion or peptone), yeast extract (Preferably fresh) and horse serum (10%). Several other components can be added, such as glucose, glycerol, DNA and fatty acids, but the effects vary with the strains [1]. To avoid growth of other bacteria, inhibitors, such as penicillin, colistin or thallium acetate, are necessary. The media can be used as broth or solid medium with 1.0–1.2% agar [2].

All culture media prepared should be subjected to quality and must support growth of *Mycoplasma* spp. from small inocula. The reference strain should be cultured in parallel with the suspicious samples to ensure that the tests are working correctly. After grinding in broth containing antibiotics, the lung samples are diluted tenfold to minimize contaminating bacteria and are inoculated into five tubes of broth and on to solid medium. The pleural fluid can be inoculated directly without previous dilution. Hermetic sealing of the Petri dishes or the uses of incubators with controlled humidity are recommended in order to avoid desiccation. To ensure the best conditions for mycoplasma growth, a CO_2 incubator or candle jar should be used. The tubes and petri dishes are inspected at day 5 and at day 10. In fluid medium, a homogeneous cloudiness usually appears within 2–4 days, frequently with a silky, fragile filament called a ‘Comet’, which is characteristic of (Or *M. capricolum* subsp. *capripneumoniae*, the cause of contagious caprine pleuropneumonia). During the following days a uniform opacity develops with whirls when shaken. On agar media, the colonies are small (1mm in diameter) and have the classical appearance of ‘fried eggs’ with a dense centre. At this stage, the indirect fluorescent antibody (IFA) test or PCR can be performed [1].

Biochemical Tests: For routine field use, the immunological tests and PCR are sufficient, but where these give dubious results, biochemical tests may be used. These biochemical tests should be carried out by a reference laboratory [31]. For this purpose, after two or three subcultures, antibiotics should be omitted from the medium to check if the isolate is a mycoplasma or an L-form of a bacterium that will regain its original form in the medium without inhibitors. Once this test is done and after cloning (At least three colonies should be selected), the organism can be identified using biochemical tests [34].

Mycoplasma mycoides subspecies *mycoides* Small Colonies is sensitive to digitonin (Like all members of the order Mycoplasmatales), does not produce ‘film and spots’, ferments glucose, reduces tetrazolium salts (Aerobically or anaerobically), does not hydrolyse arginine, has no phosphatase activity and has no or weak proteolytic properties. For these tests, special media have been developed that include the same basic ingredients (Heart-infusion broth or Bacto PPLO [Pleuropneumonia-like organisms] broth, horse serum, 25% yeast extract solution, 0.2% DNA solution), to which is added 1% of a 50% glucose solution for glucose hydrolysis, 4% of a 38% arginine HCl solution for arginine hydrolysis and 1% of a 2% triphenyltetrazolium chloride solution for tetrazolium reduction, plus a pH indicator (e.g. phenol red). (Note: a pH indicator should not be added to a medium containing triphenyltetrazolium chloride). For demonstration of proteolysis, growth is carried out on casein agar and/or coagulated serum agar [1, 34].

Once the biochemical characteristics have been checked, one of the following immunological tests can be performed to confirm the identification: disk growth inhibition test (DGIT), fluorescent antibody test (FAT) and the dot immunobinding on a membrane filter (MF-dot) test. The isolation and identification of the CBPP agent can be difficult and time consuming and depends on careful of the appropriate procedures and media. When possible, classical bacteriology laboratories should set up a special section for work only with mycoplasmas [31].

Serological Tests: Serological tests for CBPP are valid at the herd level only. Tests on single animals can be misleading, either because the animal is in the early stage of disease, before specific antibodies are produced, or it may be in the chronic stage of the disease when very few animals are seropositive [33].

Complement fixation (A test suitable for determining freedom from disease and a prescribed test for international trade): The Campbell & Turner complement fixation (CF) test remains the recommended procedure (Although the current method is slightly different from the original one) and it widely used in all countries where infection occurs [25].

It is recommended that any fixation of complement, even partial (25, 50 or 75%), at a serum dilution of 1/10 should be followed by additional investigations. The limitations of the CF test are well known. With a sensitivity of 70% and a specificity of 98% (7), the CF test can detect nearly all sick animals with acute lesions, but a rather smaller proportion of animals in the early stages of the disease or of animals with chronic lesions. In addition, therapeutic interventions and improperly conducted prophylactic operations (Partial slaughter of the herd) may increase the number of false-negative reactions. However, for groups of animals (Herd or epidemiological unit) the CF test is capable of detecting practically 100% of infected groups. The nature of the pathogenesis of the disease is such that the incubation period, during which antibodies are undetectable by the CF test, may last for several months. Despite the high specificity of the CF test, false-positive results can occur, of which an important cause is serological cross-reactions with other mycoplasmas, particularly other members of the *M. mycoides* cluster. The validity of the results has to be confirmed by post-mortem and bacteriological examination and serological tests on blood taken at the time of slaughter [35].

Competitive enzyme-linked immunosorbent assay (A prescribed test for international trade): A competitive enzyme-linked immunosorbent assay (C-ELISA) developed by the OIE Collaborating Centre for the diagnosis and control of animal diseases in tropical countries has undergone evaluation [1, 13].

An indirect ELISA based on the use of a lipoprotein antigen is currently being validated by the IAEA. In May 2004, the c-ELISA was designated as an OIE prescribed test for international trade by the OIE International Committee. Compared with the CF test, the c-ELISA has equal sensitivity and greater specificity. Advice on the availability of reagents can be obtained from the OIE Reference Laboratories for CBPP or the OIE Collaborating Centre for ELISA and Molecular Techniques in Animal Disease Diagnosis Validation tests that have been carried out in several African and European countries would indicate [13] that (1) the true specificity of the c-ELISA has been reported to be at least 99.9%; (2) that the sensitivity

of the c-ELISA and the CF test are similar and (3) antibodies are detected by the c-ELISA in an infected herd very soon after they can be detected by the CFT and c-ELISA antibody persists for a longer period of time [36].

The c-ELISA is now provided as a readymade kit that contains all the necessary reagents including precoated plates kept in sealed aluminum foil. The kit has been especially designed to be robust and offer a good repeatability. As a consequence, sera are analyzed in single wells. The substrate has been modified and is now TMB (Tetra methyl Benzedrine) in a liquid buffer and the reading is at 450 nm. The substrate color turns from pale green to blue in the first place and becomes yellow once the stopping solution has been added. Monoclonal antibody (MAb) controls exhibit a darker color while strong positive serum controls are very pale. The cut-off point has been set at 50% and should be valid in every country [1].

Immunoblotting Test: An immunoenzymatic test designated the immunoblotting test (IB test) has been developed and is of diagnostic value. A field evaluation indicated a higher sensitivity and specificity than the CF test. A core profile of antigenic bands, present both in experimentally and naturally infected cattle are immunodominant. The more accurate picture of the immune status of animals given by this test is due to the possibility of a more precise analysis of the host's immune response in relation to the electrophoretic profile of *MmmSC* antigens; thus the test overcomes problems related to nonspecific binding. It should be used primarily as a confirmatory test, after other tests and should be used in all cases in which the CF test has given a suspected false result [1, 32].

Nucleic Acid Recognition Methods: Radio labeled or enzyme probes have been developed, but have been superseded by the more convenient and safe PCR technology. The PCR is sensitive, highly specific, rapid and relatively easy to perform. Primers specific for the *M. mycoides* cluster and *MmmSC* have been reported and PCR assays have been developed, including a new technique that permits the identification of the T1 vaccinal strains [37]

Using samples such as lung exudates allows the PCR to be performed directly after differential centrifugations to remove inflammatory cells and pellet mycoplasmas. For fragments, the PCR is applied after DNA extraction. The PCR can also be performed on urine or blood. The main advantage of the PCR technique is

that it can be applied to poorly preserved samples (Contaminated or without any viable mycoplasmas as may occur following antibiotic treatment). If direct detection of DNA from the organ under test fails, specimens should be enriched by culturing them in an appropriate medium for 24–48 hours, followed by attempted detection of DNA from culture [1, 37].

The PCR has become the primary tool for identification of *MmmSC*. If a sample is PCR positive in a CBPP-free zone, the test confirmed by a second and different PCR; infection can be confirmed by the use of only one immunological test. One of the problems with PCR is the possible occurrence of contamination if the necessary precautions and quality management system are not implemented correctly in the diagnostic laboratory. Great care must be taken to respect the strict separation between those parts of the laboratory that may be contaminated with PCR products (Such as the electrophoresis room) and those parts of the laboratory devoted to preparing the reagents [31].

Differential diagnosis: In carrying out a CBPP diagnosis, it is necessary to differentiate this disease from other diseases which may present similar clinical signs or lesions. The way the disease behaves in the herd is as important as the findings in a single animal when carrying out an investigation. The following diseases should be considered in differential diagnosis of CBPP [2, 9, 10, 31].

Rinderpest: The confusion with rinderpest results from the fever and discharges observed from the eyes, nose and mouth. However, the characteristic lesions of rinderpest those are essentially erosions in the mouth and throughout the digestive tract, together with the profuse, often bloody, diarrhoea in advanced cases, should enable easy differentiation from CBPP in which these are not seen. Lung lesions are seen in more chronic cases of rinderpest and these consist of red areas of collapse together with emphysema of lung lobules and the septa separating them. At this stage the erosive lesions of rinderpest may have healed [31].

Foot-and-mouth Disease: Salivation, lameness and fever are the cause of confusion [21].

Haemorrhagic septicaemia: This is a very acute disease and most affected animals die within 6 to 72 hours after the onset of clinical signs. Buffaloes are particularly susceptible. Oedema of the throat and neck to the brisket is often very pronounced. The lung lesions seen in

animals that survive the longest can appear very similar to the marbling lesion of CBPP, there may be yellow fluid in the chest and the affected lung may adhere to the inside of the rib cage. Thus, in the individual case distinguishing between HS and CBPP can be difficult [31].

Bacterial or Viral Bronchopneumonia: Clinical signs may resemble closely those of acute CBPP. Post-mortem examination shows usually both lungs to be affected, fibrinous exudates may be present but not to the same extent as in CBPP. While dark, solid areas of lung may be seen, these are usually restricted to the anterior lobes (not the diaphragmatic lobe as in CBPP) and marbled lungs are not often seen [9].

Theileriosis (East Coast Fever): Coughing, nasal and ocular discharge and diarrhea are observed. Affected cattle show general enlargement of superficial lymph nodes and especially those of the head. The lungs contain much clear liquid which is also present in the chest cavity; the airways in the lung may be filled with white froth. Cigarette urn-like ulcers are seen in the abomasal folds. Neither pneumonia nor inflammations of the pleura are present [20].

Ephemeral Fever: In most cases this is a self-limiting disease of short duration; most affected cattle recover quickly, even those which are severely affected. The fever fluctuates with two or more peaks. Pneumonia is not a main feature of the disease but a secondary pneumonia can occur with lung oedema and emphysema in a small proportion of cases. Confusion with CBPP arises from the presence of fever, discharges from the eyes and dripping of saliva from the mouth, lameness and swollen joints (But in animals of all ages unlike CBPP) [32].

Abscesses: They can be mistaken for sequestra. When cut open the contents of abscesses are seen to be offensive smelling, liquid purulent material, absent in sequestra. In abscesses a total destruction of the lung tissue occurs. Old thickly encapsulated hydatid cysts can also cause some confusion [2].

Tuberculosis: Tubercular nodules can superficially resemble sequestra but they are degenerative cheese-like lesions, sometimes calcified. The lung tissue is destroyed and the same lesions are also seen in lymph nodes in the chest. The capsule of the tubercular nodules is not well defined when compared to that of sequestra [9].

Farcy: The lung lesions of farcy differ from sequestra as they are filled with foul smelling purulent material (Same as abscesses). Similar lymph node lesions are always present [31].

Actinobacillosis: The pulmonary lesions, when found, could be mistaken for sequestra. Lesions are generalized and seldom present in lungs [24].

Echinococcal (Hydatid) Cysts: These cysts having a double wall and contain a clear liquid, often calcified when old [2].

Foreign Body Reticulum Pericarditis: Mostly one animal is affected. The two diseases could be clinically misunderstood, but not epidemiologically and pathologically [31].

Treatment- Under practical field conditions, when the disease out breaks in a new area, treatment is not applicable and not recommended because of reasons of disease prevention (Seifert, 1996). Treatment is usually undertaken and indicated only in areas where the disease is endemic [10], but in practice farmers are treating their animals when they have no other alternative. Although the Mycoplasmas are susceptible to a number of antibiotics *invitro*, treatment failures are common [21]. Commonly used antibiotics include tetracyclines, tylosin, erythromycin, lincomycin, spectinomycin and tilmicocin [21]. Tyrosine and spiramycin are effective in the control of excessive vaccination reactions and should be of value in the treatment of clinical cases. Resistance to some of these antimicrobials has been noted. Animals that do not respond to treatment often become carriers. Penicillin is of little value, streptomycin has some curative effect [9, 10].

Control and prevention- To make the most efficient use of the increasingly scarce resources, disease control programs must be tailored to the needs of particular communities and to high-priority cattle populations to ensure their efficacy, acceptance and sustainability and therefore economic evaluation should be generalized [20]. The major obstacles to the control and eradication of the disease are: difficulty in controlling of animal movements, especially in sub-Saharan Africa, complications of applying quarantine and slaughter policies, lack of rapid pen side diagnostic test, in effective vaccine and in sufficient funds to implement control policies [9]. A variety of management options exists when local, national or international authorities face decisions on

transboundary animal disease, like CBPP [38]. Control of animal movement (Quarantine and isolation): testing of suspected animals, slaughtering those infected and disposing of carcass by burial or burning (Stumping out), vaccination of susceptible animals [27].

Stamping- Out: The ideal method to control a transboundary disease like CBPP is the application of the stamping out policy of complete elimination of infected and exposed animals along with attendant zoo-sanitary measures. This strategy is generally design to for slaughtering of animals during the epidemicity of the disease to reduce the risk of transmission [20, 39].

This policy will probably be most important for countries with highly developed livestock industries. It involves the irradiation of disease by distraction of all infected animals [32]. It should not be contemplated unless there are adequate provisions for compensation. If there is no any compensation for stumping out, then producers, particularly small scale producers are reluctant to participate and if they participate it may mean that no longer can afford to produce. In order to avoid decapitalization, small scale producers who rely on solely on their animals for income may move their animals across the border rather than killing them, farther spreading infection [32].

Test and Slaughter Infected Animal: In eradication campaign, infected animals may be slaughtered to remove source of infection. Eradication of a disease from herds after involves a test and removal strategies, in each all animals are tested and only those positive are removed and slaughtered [39].

Quarantine: Uncontrolled animal movements during transhumance, trade and cattle theft have facilitated the spread of the disease throughout the world. Although the quarantine and checkpoints have been in place, weak legislation and a lack of means and resources to enforce control of livestock movements are making the situation worse [40]. Then this is strategy for isolation of animals that are either infected or suspecting of being so, or of non infected animals that are at risk. It is also important to isolate animals suspected of being infected, until infections is either confirmed or discounted by clinical examination or laboratory testing. Within each quarantine areas, clinical cases are separated and confirmed to a hospitalized zone and such animals are slaughtered under strict veterinary supervision [39].

Vaccination: Where the application of the stamping out policy of eradication is not feasible the control of CBPP has relied on preventive immunoprophylaxis using live attenuated cultures of the causative agent along with restriction of cattle movement if possible [25]. CBPP vaccination is the method that is currently in use in most African countries employing the vaccine strains T₁/44 or its streptomycin resistant derivative T₁-SR for a long time, liquid culture vaccine were successfully used to control CBPP especially in East Africa and Australia[41].

Economic Importance of CBPP: CBPP is considered to be a disease of economic importance because of its high mortality rate, production loss, increased production cost due to cost of disease control, loss of weight and working ability, delaying marketing, reduced fertility, loss due to quarantine, loss of cattle trade, reduced investment in livestock production[3, 9]. In addition to these, it leads to in imposition of rigorous limitations to international trades on CBPP-affected countries in accordance with world organization of Animal Health (OIE) regulations [7, 42].

The financial and economic loss caused by the disease in Africa is significant. Otte *et al.* [38] reported that the continent has lost approximately 2 billion US dollar per year due to death of livestock from the disease. Contagious bovine pleuropneumonia has been causing significant economic loss on the agriculture sectors and the national economy. It accounts for a loss of over 206.5 million Ethiopian birr per year [43]. Thus, over the last decades, the country has lost a substantial market share and foreign exchange earnings due to frequent bans by the Middle East countries [19].

The Epidemiology of Cbpp in Ethiopia: The origin of CBPP in Central, West and East Africa is obscure and it has been suggested that the infection was introduced by zebu cattle when they first migrated to the African continent. There is a suggestion that CBPP was introduced into East Africa from India by the army of field Marshal Napier when he invaded Ethiopia in 1867-1868 [26], while Tulasne *et al.* [44] have reported that the traditional practice of provoking "Willems reaction" was rediscovered by Willemms in 1854. This indicates that CBPP had existed in Africa before 1854 [44].

After Rinderpest has been brought under control, CBPP is considered to be among the most important cattle diseases and impediments to livestock development in Ethiopia, particularly in the lowlands. CBPP is one of the

great plagues which continue to devastate cattle herds on which so many people are dependent in the lowlands. In the highlands, the consecutive yearly blanket vaccinations with combined Rinderpest and CBPP have certainly contained the disease to a relatively low level during the past years. But with the adoption of a strategy towards Rinderpest eradication, the vaccinations in the highlands have ceased since 1992/93 [15].

Generally, the irregularity and low rate of vaccinations since 1993 seem to contribute to the increased incidence of the disease and its further spread. The usual blanket coverage was around 50% and never reached the desired 80-100% level [15].

According to eleven years (1992 – 2002 G. C.) disease outbreak reports by Federal Ministry of Agriculture, several CBPP epidemics have been recorded from the south, south-west, west, north-west and north-east regions of the country. The passive disease outbreak reports from 1992-2002 shows 587 outbreaks, 16,806 cases and 3,262 deaths. The highest record was in 1998 when 187 outbreaks with 5,652 cases and 1071 deaths were reported [45]. However, this data cannot be used to determine, the level and geographic feature of the disease, determine the importance of the disease, set priorities for the use of resources for disease control activities, plan, implement and monitor diseases control program, or demonstrate disease status to trading activities. Due to the insidious nature of the disease, such official data do not necessarily convey the extent of the problem caused by CBPP in Ethiopia [18].

Studies under taken on CBPP so far revealed the existence of the disease in different parts of Ethiopia with the prevalence of that vary from 43% in Jijiga [16] to 96% in western Gojjam [17]. Studies conducted in Western Ethiopia [46, 47], Northwest Ethiopia [48], Southern Ethiopia [49] and different regions of the country [50] revealed that CBPP is posing a major threat to cattle in many parts of the country thereby causing considerable economic losses through morbidity and mortality and warranting for serious attention [18]. The cattle population at risk of CBPP and livestock production systems in CBPP endemic and epidemic zones of Ethiopia is estimated to be a total of 13,325,700 heads of cattle. All of them are considered to be at risk of CBPP, of which 5,510,700 are in endemic zones and 7,815,000 are in epidemic zones. Generally, based on the available information, the epidemiological situation of CBPP is found in various parts of Ethiopia (Fig. 1) [18]:

Table 1: Cattle population at risk in 4 CBPP affected areas of Ethiopia

Area	Administrative Zones	Cattle population	Livestock system
Western Ethiopia	Endemic Zones		Mixed crop-livestock
	- Western Wellega (Oromia)	1 005 500	
	- Asosa (B. Gumuz)	84 200	Mixed crop-livestock
	Epidemic Zones		
	-Part of W. Wellega (Oromia)	272 700	Mixed crop-livestock
North Western Ethiopia	Endemic Zones		
	- Western Gojam (Amhara)	1 188 000	Mixed crop-livestock
	- Awi (Amhara)	470 000	Mixed crop-livestock
North Eastern Ethiopia	Endemic Zones		
	- Afar Zones (Afar)	768 000	Nomadic
	Epidemic Zones		
	- southern Tigray (Tigray)	450 000	Mixed crop-livestock
	- North Wello (Amhara)	620 000	Mixed crop-livestock
	- North Shoa (Amhara)	1 018 000	Mixed crop-livestock
	- Eastern Shoa (Oromia)	1 019 000	Mixed crop-livestock
	- Arsi (Oromia)	2 509 000	Mixed crop-livestock
Southern Ethiopia	Endemic Zones		
	- Borena (Oromia)	1 419 000	Nomadic
	- South Omo (SNNP)	413 000	Mixed & nomadic
	- Konso S.D. (SNNP)	70 000	Mixed crop-livestock
	- Derashe S.D. (SNNP)	34 000	Mixed crop-livestock
	- Amaro S.D. (SNNP)	59 000	Mixed crop-livestock
	Epidemic Zones		
	- North Omo (SNNP)	1 715 000	Mixed crop-livestock
	- Maji (SNNP)	212 000	Mixed & nomadic
Total	Endemic zones	5 510 700	
	Epidemic zones	7 815 000	

Source: Cattle population in the Zones: CSA[51], Livestock and Poultry and beehives population

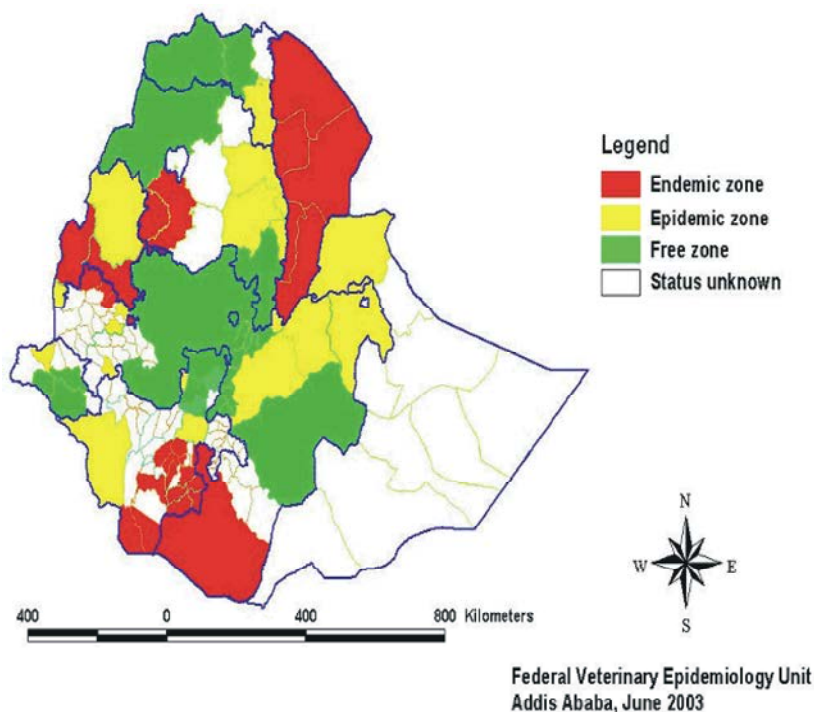


Fig. 1: Map showing the different CBPP zones in Ethiopia
Source: Federal Veterinary Epidemiology Unit Addis Ababa [52]

Western Ethiopia including Western Wollega and Assosa Zones (And possible a part of Gambella Region) are considered endemic and epidemic; Southern Ethiopia: (Southern Nation, Nationalities and People region, SNNPR). Borena Zone as a whole (Oromia Region), infected since long time, is an endemic area and characterized by pastoralism; South Omo, Konso Derashe and Amaro Zones (SNNP Region) are considered as endemic, with recent outbreaks in the neighboring Zones such as Bench Maji and North Omo Zones; Gondar and Gojam areas have declared numerous outbreaks since 1993 and South Gondar and West Gojam are categorized as epidemic areas. West Gojam zone comprises of seven districts, namely Burie-Wonberma, Denbecha, Jabitenan, Dega-Damot, Quarit, Sekela and Achefer, of which the first two districts are considered CBPP endemic and the last four districts are considered CBPP free; the highlands of North Shewa were considered as CBPP free, however, Wondimu [49] reported sero-prevalence rate of 54% using CFT; Southern Tigray seems to be recently infected with sero-prevalence rate of 50% reported in 1996 [49] and this can be categorized as epidemic; AgewAwi zone comprises of four districts, namey Dangela, Ankeshaguagusa, Gungua and Banja-shikudad, of which the first three are considered as CBPP endemic and the last is with sporadic occurrence (Table 1). Here mixed crop-livestock production system is practiced and the dominant livestock species are cattle; North Eastern Ethiopia: Afar Region as a whole and Northern Somali Region may be considered as endemic, with recent outbreaks encroaching on the edge of endemic are in Southern Tigray, North Wello, North Shewa, East Shewa, (Amhara Region) and Arsi Zone (Oromia Region); Eastern Ethiopia: In Somali Region except one zone, Shinille, which is considered to be CBPP epidemic zone, the status of the disease in all the other zones is unknown. Once introduced to a new area, initial losses in pastoral communities can be very high and its eradication is very difficult requiring major expenditure for control [49].

The Cbpp Diagnosis in Ethiopia: Epidemiological investigation to obtain a general picture of the way the disease has behaved in the herd; clinical examination: how the animals of a herd are affected by the disease; Post-mortem examination to observe the characteristic lesions in organs of dead and/or slaughtered animals; laboratory examination to confirm the presence of infection [16].

An outbreak of contagious bovine pleuropneumonia (CBPP) was investigated in the Somali National Regional State, Eastern Ethiopia, to isolate and identify the

causative *Mycoplasma* species. Sick animals for autopsy and bacteriological specimen collection were out looked. Postmortem examination and sample collection was performed on 7 recently dead animals. The clinical and pathological findings encountered and the bacteriological as well as the biochemical tests performed, established the outbreak to be CBPP [53].

Clinical and Necropsy Findings: Clinical examinations of infected animals revealed nasal discharge, coughing, labored breathing, disinclination to move and postures that showed the animal was fighting to get enough oxygen. The profound lesions observed on postmortem showed adhesion of the pleura with the chest wall and the lung and consolidated lung tissues with characteristic marbling. The pleural cavity was full of copious, yellowish-colored clear fluid. Heavy deposits of fibrin flocculates were encountered [53].

Bacteriological Findings: Evidence of the growth of *Mycoplasma* organisms was based on a change in color of the growth medium from pink to yellow. Moderate turbidity with a whitish deposit at the bottom of the culture vessels were additional parameters used to determine *Mycoplasma* growth. Both the tissue sample processed and the pleural fluid cultured were positive for *Mycoplasma* growth after incubation for 72–120 hours in broth culture media. Gram-stained smears from these cultures showed the presence of gram-negative, pleomorphic organisms composed of coccoid, coccobacillary and filamentous organisms. Giemsa-stained preparations from the culture suspensions revealed coccoid, pear-shaped, and filamentous microorganisms. Growth on solid medium was characterized by the presence of microcolonies with a typical nipple-shaped appearance after 7 days of incubation. The colonies were observed under inverted microscope (32X) with transmitted light. **Biochemical and Biological Properties:** Positive results were seen for growth in aerobic conditions, glucose fermentation, digitonin sensitivity, ability to pass through a 0.45- μ m membrane, growth inhibition and sensitivity to chloramphenicol and tetracycline. Negative results were seen for arginine hydrolysis, urea breakdown, growth on serum-free media and sensitivity to penicillin [16, 53].

Serological Tests: The complement fixation test on serum is still the most useful methods of detecting infection. It is a rapid, simple and easy to perform and interpret the results. It is more specific than ELISA tests.

It lacks sensitivity for serum samples having a very low antibody level. ELISA tests detect late and persistent infections while CFT detects early infections [54].

CBPP Control Methods (Strategies) in Ethiopia: The major control method practiced in Ethiopia is vaccination. The control of CBPP by vaccination has been carried out for the last 30 years. Previously consecutive yearly blanket vaccination with combined Rinderpest and CBPP vaccine was adopted as a strategy to control CBPP. It was this strategy that is believed to have contained the disease to a relatively low level until 1992/93. And this method was considered as a successful achievement in the control of CBPP. However with the adoption of a strategy towards Rinderpest eradication, the vaccinations in the highlands and most parts of the Somali region have ceased since 1992/93. Besides, the vaccination coverage was around 50% and did not reach the desired 80 – 100% level. Currently, CBPP control in Ethiopia is based on targeted and ring vaccination in the face of outbreaks [55].

CONCLUSIONS

Contagious bovine pleuropneumonia is highly contagious disease of cattle caused by *Mycoplasma mycoides subspecies mycoides* SC type. The disease is found in different parts of the world; especially it is the problem of developing countries, Ethiopia due to lack of enough diagnostic tools, well trained personnel, economy, strategic epidemiological surveillance for the eradication of the diseases. The epidemiological situation of the disease is found in various parts of Ethiopia. It is an endemic disease in most parts of the country. Contagious bovine pleuropneumonia is posing a major threat to cattle in many parts of the country thereby causing considerable economic losses through morbidity and mortality. Diagnosis of the disease in Ethiopia is performed through clinical finding, necropsy finding, culturing and serological technique. The main control strategy in Ethiopia is done through vaccination; and sometimes control is done by restricting the movement of cattle. However, vaccination cover is usually not very high, due to financial and government policy constraints. Therefore, based on the above conclusions, the following recommendations are forwarded.

- Adequate funding should be available to control CBPP in Ethiopia, as well as other countries.
- Research in the improvements of vaccine should continue and include the possibility of differentiation between vaccinated and non-vaccinated animals.

- Veterinarian should aware of the pastoralist about the problem of the disease.
- Strategic control of CBPP should be progressive and based on impact assessment and cost benefit analysis done with appropriate methods including participatory techniques to cover regional, national, zone level.
- A veterinarian should well trained to perform diagnosis of the disease through the available diagnostic tools.
- Research should be done on the epidemiological situation of the disease

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